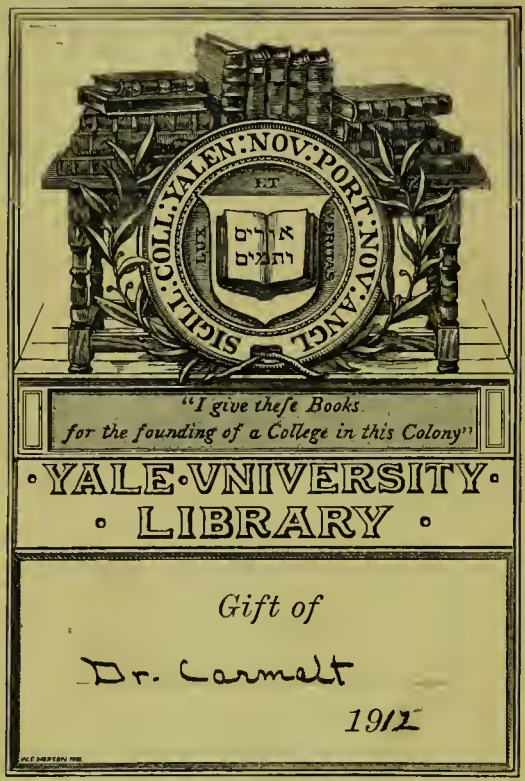
ON

TUBERCULOSIS

DR HORACE DOBELL.



TUBERCULOSIS.

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TUBERCULOSIS:

ITS

NATURE, CAUSE, AND TREATMENT.

WITH NOTES ON PANCREATIC JUICE.

BY

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ETC. ETC.

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PREFACE

TO THE

SECOND EDITION.

My publishers having called upon me to prepare a second edition of this book within two months of its first publication, I cannot be expected to make any substantial alterations or additions; but I have carefully revised the text, and inserted a few notes.

41, Harley Street,
April, 1866.

INTRODUCTION.

The following papers appeared at distant intervals in the columns of different journals; and the reports of the experiments were published before the statement of the views which led to their adoption. I have now collected the several papers, and placed them in their proper order, for the convenience of those who may be interested in the important subject to which they relate.

Several curious scientific phenomena have been observed by Mr. Heathorn, Mr. Schweitzer, Mr. Morson and myself, while acting upon fats and other bodies with pancreatic juice; and I hope that, as soon as these observations have assumed a less crude form, one of these gentlemen will be induced to collect them and make them public. I think it will then be shown that many inaccuracies exist in the accounts of the physiology and chemistry of the pancreatic juice at present published.

The most useful treatise on the pancreas and its

diseases with which I have met, is that written by Dr. Copland, about twenty years ago, and published in his Dictionary of Practical Medicine. In this he makes some most important suggestions as to the probable existence of functional disorders of the pancreas influencing the quantity of its secretion, and thus producing serious derangements of the digestive processes. I would especially refer those who are interested in the subject to this treatise, and also to a work of Dr. T. King Chambers ("Digestion, and its Derangements"), published in 1855, in which the influence of the pancreas on digestion is discussed at some length.

The physiology of the pancreas has long excited attention, and has led to considerable differences of opinion. Majendie, Tiedemann, Gmelin, Lassaigne, Leuret, and others pointed out an analogy between the effects of saliva and of pancreatic juice in the digestion of starch. The operation of the pancreas in the digestion of nitrogenous matters has been principally made known by the persevering experiments of M. Lucien Corvisart, published at various times from 1857 to 1863. But our knowledge of the important functions of this organ in preparing fats for assimilation, is almost entirely due to the experiments and researches of Dr. Claude Bernard, first published in the Archives Générales de Médecine, 1849, and followed up by him ever since. I am glad of this opportunity of

expressing how much I owe to the experiments of Professor Bernard, for assistance in my own investigations into the assimilation of fat in consumption.

In 1858, Dr. G. Harley read a paper to the British Medical Association ("Notes of Experiments on Digestion"), in which he gave an interesting *resumé* of the knowledge possessed at that time of the physiology of the pancreas, and he expressed an opinion that "as a remedy in indigestion, pancreatine should be greatly superior to pepsine," because, as shown by Bernard, Corvisart, and others, it unites in itself the properties of all the other digestive fluids. This paper will well repay perusal.

In Dr. Bright's celebrated observations, published in the Medico-Chir. Transactions, 1833, he attempted to establish the fact that the excretion of undigested fat by the bowels was a constant accompaniment of obstruction to the pancreatic duct. A doubt was afterwards cast upon the validity of his conclusions, from the circumstance that the only remaining specimen of the "fat" passed in his cases, preserved in the Museum of the Royal College of Physicians, was examined in 1850, and found not to be fat at all, but a piece of undigested meat.

But there is no doubt that this was only an unfortunate accident. Dr. Bright was far too astute an observer to make such a foolish mistake in a series of cases.

I have proved for myself, in one case, at least, that undigested fat may be found in the stools of a person suffering from obstruction of the pancreatic duct. In 1862 I was consulted about a patient of Dr. A. A. Davis, of Fowey, Cornwall, who was passing in his motions pellets of hard whitish matter, which had for some time attracted his attention. These were analysed and examined microscopically by my friend Mr. Farrants, late President of the Microscopical Society, and myself, and found to consist of pure stearin: the albuminoid cells of the adipose tissue and the olein had been removed, leaving nothing but hard masses of stearin to be excreted. From this and other symptoms I gave a positive diagnosis of disease of the pancreas. The patient subsequently died, and the pancreas was examined by Dr. Andrew, of St. Bartholomew's Hospital, and myself, when the large end of the organ was found to be enlarged, the duct obliterated and imbedded in a mass of cancerous deposit. An attempt was made in this case to prolong life by partially feeding the patient upon calf's sweetbread, no satisfactory process being then on record by which to obtain the active principles of the pancreatic secretion.

At the time I attended this patient I was planning my experiments on the assimilation of fat in consumption, which I commenced during the following year at the Royal Infirmary for Diseases of the Chest, and the

first difficulty that I had to overcome was to discover some means of obtaining the active principles of the pancreatic juice, and some form in which to administer them as a remedy. At that time it was found, on enquiring for assistance in this matter, that neither pancreatine, pancreatic juice, nor any preparation from it had ever been heard of as remedies by the most celebrated chemists in London, who are called upon to prepare all the rarest medicaments, and to dispense prescriptions and receipts from all parts of the world. Messrs. Savory and Moore, Allen and Hanbury, Bell, Morson, and others, although, as scientific chemists, acquainted with the physiological properties of the pancreatic secretion so far as then known, had none of them heard of its use in medicine.*

Being unable, therefore, to get any useful information on the subject, either from books or from practical men, it rested with me to solve the difficulty. At this point in my experiments I obtained the assistance of Mr. Heathorn, and great credit is due to him for the perseverance with which he

* Plough-Court, Lombard-street, E.C.
London, Dec. 14, 1865.

Dear Sir,—We never had an enquiry for Pancreatic juice or any preparation from it, before the time when you introduced the *Pancreatic Emulsion;* and we are not aware of Pancreatic juice having been previously prescribed in any form. We remain, dear Sir, yours faithfully, ALLEN AND HANBURYS.

Dr. Dobell.

applied himself to the troublesome task. The problem was not an easy one, but it was at length solved, and the "Pancreatic Emulsion" was the result. We found that there are several active principles of the pancreatic secretion all essential to its proper function. *There is none of these which taken singly completely represents the true properties of healthy pancreatic juice.* I consider the pancreatic emulsion of solid fat prepared with the entire juice of the pancreas of a pig just killed, to be the most reliable form in which to use the remedy. Since Mr. Heathorn ceased to make the pancreatic emulsions, many chemists have submitted specimens to me for examination, but, up to the present time, I have not seen any to which I could give my unqualified approval, except those prepared by Messrs. Savory and Moore.

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TUBERCULOSIS:

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NATURE, CAUSE, AND TREATMENT.

[Reprinted from the British Medical Journal, Jan. 20 and Feb. 10, 1866.]

THE greater part of my leisure during the last sixteen years has been devoted to a study of the natural history of pulmonary consumption and other forms of tuberculosis, and to collecting, assorting, comparing, and testing the facts recorded by others directly and indirectly connected with the subject.

The ideas which led to this plan of work were set forth in a paper which I read to the Abernethian Society of St. Bartholomew's Hospital in 1848, and afterwards published in the *London Medical Gazette* of 1851, "On the Class of Medical Literature most needed in the Present Day."* The object was to

* These papers, as corrected and reprinted in 1857, may be obtained from Messrs. Churchill.

bring together in an epitome all the most reliable facts concerning tuberculosis, and, from a careful study of them as a whole, to frame hypotheses which seemed capable of co-ordinating and explaining large numbers of the principal facts; then to select the one which included the largest number, and to contrive some crucial experiments to test its correctness.

This plan of procedure was carried out, with the effect of annihilating one hypothesis after another, each time suggesting some apparently better arrangement of the facts and some more appropriate ideas with relation to them, until, at length, one hypothesis, with certain possible variations, appeared to grow nearer and nearer to a complete theory.

I have again and again been on the point of publishing this view of the causes of tuberculosis; but have hesitated from a feeling that, before doing so, it ought to be brought up to the completeness of a theory; and my attempts to give it this completeness having always failed to satisfy me, I have been thus deterred from bringing it forward. I have, however, gradually become impressed with the belief that this incompleteness may have been due in some measure to my own want of a sufficiently profound knowledge of physiology and organic chemistry, and

of the leisure necessary to acquire this knowledge and to carry out experiments in physiological chemistry. I have hoped to engage the assistance of those whose acquirements in this respect are better than my own, and who, being less occupied with the practice of medicine, have more time for the required class of work. In this hope I have been, at present, disappointed, both because the number of such men is limited, and because they have their heads and hands preoccupied with their own studies, and are naturally unwilling to leave these and apply themselves to such as may be suggested by other persons.

The reasons which have deterred me so long would deter me still from venturing to bring my hypothesis before the profession, were it not that having, during the last two years, published in the *Lancet* (September 10, 1864, June 10, 1865, November 11 and 18, 1865) the practical results of a crucial experiment, without giving any account of the hypothesis on which it was based, and these results having attracted the attention of my medical and scientific brethren, and given rise to many inquiries, I find myself called upon, in simple courtesy, to give some answer to these inquiries. Under these circumstances, and as the views which

I entertain, whether they prove true or not, are at least the result of an inquiry conducted on philosophical principles, I feel that I cannot do better than lay them before the profession for discussion.

The hypothesis may be briefly stated as follows.

Tuberculosis is due to defect in the action of the pancreas on the fat taken as food (especially the solid fat). The supply of properly prepared fat is cut off from the blood: 1, by the fats not being brought into a proper condition by the pancreas; 2, by loss of absorbing power in the small intestine, due to the contact of unhealthy pancreatic juice and of defectively prepared food with its mucous membrane. Thus, the blood becomes deficiently and defectively supplied with fat-elements from the food; is unable to afford those required for direct combustion; does not replace those taken up during interstitial nutrition; but, on the contrary, takes up more to compensate the deficient supply from the food. This having gone on up to a certain point, the fat-elements of the albuminoid tissues * are seized upon, and these tissues are minutely disintegrated in the process. This disintegrated albuminoid tissue

* The word "tissues" is here intended to include the albuminoid materials employed in the construction and repair of tissues, for it is probable that the fat elements may be waylaid during the process of tissue formation.

is nascent tubercle; and this process of disintegration is tuberculisation.

Tuberculisation will take place first wherever the following combination of conditions is most marked.

1. Greatest activity of interstitial nutrition.

2. Smallest amount of fat-elements able to be spared by the tissues.

3. A double process going on, consisting of: (*a*) ordinary interstitial nutrition in albuminoid tissue; (*b*) interchange of oxygen and carbonic acid or carbonaceous matters through this tissue.

The tubercle thus formed may be allowed to remain on the spot where it is formed, constituting a *primary deposit at the point of origin*, and this will especially occur when formed under the three conditions just specified; or, it may not be allowed to remain on the spot where formed, but be at once carried away by the lymphatics in a minutely divided condition, and either arrested in the lymphatic glands or carried on into the blood to be deposited from it, constituting a *primary deposit distant from the point of origin;* or, having been primarily deposited in either of these ways, it may be taken up by the lymphatics and deposited in the lymphatic glands, or carried into the blood and deposited from it, constituting a *secondary deposit.*

In the *advance* of these diseased processes, any part of the body in which nutrition is going on in albuminoid tissue may become both the source and seat of tubercle; and any part, whether albuminoid or not, if supplied with lymphaties or blood-vessels, may become the seat of tubercle.

Pure tuberculosis commences when fats, properly acted upon by the pancreas, cease to pass in normal proportions into the blood.

Tuberculisation commences when albuminoid tissue is abnormally seized upon for its fat-elements.

A secondary state is superadded when tubercle has been carried into the circulation, which constitutes tuberculæmia or tubereulous blood-poisoning.*

Tuberculosis may stop short of the production of tubercle (tuberculisation), if the normal supply of fat properly acted upon by the pancreas, be restored to the blood before the albuminoid tissues have begun to be disintegrated, and it will not return so long as this supply is kept up to a normal standard. Tuberculosis may stop short, after the production of tubercle, if the normal supply of properly prepared fat be restored; but, in this case, the tissues must have become so poor in fat-elements that it will be

* It is not certain whether or not absorbed tuberele has a zymotic action. I suspect that it has.

difficult to escape the formation of fresh tubercle in the defective tissues, while the reconstruction of normal tissue is going on. A very rapid and superabundant supply of properly prepared fat to the blood will, therefore, be required.

The balance of fat-elements in the blood and tissues having been restored, tuberculosis will cease; but tuberculæmia, with all the difficulties due to the presence of foreign decomposable and absorbable matter in the tissues and blood, may remain.

In progressing tuberculosis, after the occurrence of tuberculisation, tuberculæmia is added to and combined with the original disease. An ordinary case of consumption is a combination of these three states.

If the second portion (*b*) of the third condition specially favouring tuberculisation—viz., "an interchange of oxygen and carbonaceous matters through tissues nourished by blood deficient in fat-elements" —can be provided for by supplying carbonaceous matters to the circulating medium of the part otherwise than by the normal processes and channels, a temporary protection from tuberculisation will thereby be given to the tissues through which the interchange occurs.

If this part were previously the one in greatest danger of tuberculisation, it will for a time give pre-

cedence to the part not so protected; *e. g.* the lungs may be thus protected by the introduction of olein through the portal system, the small intestine and mesenteric glands taking precedence in the order of tuberculisation from the want of such protection.

But, as nothing can permanently protect the albuminoid tissues from disintegration except solid fat properly acted upon by the pancreas, the protection by the means above described is only temporary; and, unless the normal conditions be soon restored, the previously protected part will become tuberculous at last. By these means, however, the succession in which parts ordinarily suffer may be changed; and if the normal state be restored in time, the part temporarily protected may permanently escape disease.

The defective function of the pancreas causing defective action upon fats may be temporary or permanent; and a tendency to such defect may be hereditary.

When the normal function of the pancreas has been restored, the mucous membrane of the small intestine may not have recovered its absorbing powers; and thus the fats, though properly prepared by the pancreas, may not enter the blood.

Defect in the function of the pancreas may be produced:

1. By any cause which for a prolonged period greatly reduces its activity, by diminishing the normal demand for carbonaceous matters in the blood.

2. By the action on the nervous system of powerful or prolonged depressing influences.

3. By inflammatory and other abnormal conditions of neighbouring parts.

4. By prolonged loss of absorbing power in the small intestine by which the function of the pancreas is rendered useless.

The normal function of the pancreas may be restored (within certain limits as to the duration of the existence of the cause of its defect) by means which make a healthy demand upon its active functions; remove the depressing influences from the nervous system; and permit the absorption of properly prepared fats by the mucous membrane of the small intestine.

This hypothesis having been constructed step by step to explain the phenomena of tuberculosis, I feel safe in asserting that there is hardly a well established fact connected with the origin, course, terminations, and therapeutics, of tuberculosis, which cannot be more satisfactorily explained by this hypothesis than by any other yet suggested. I have

carefully compared all its parts with the best established views in modern physiology; and, so far as my knowledge and judgment are competent to decide, there is nothing essentially inconsistent with these views in any portion of it. At the same time, I am quite aware that there are many important points in physiology, unavoidably involved in every theory of nutrition, whether normal or abnormal, on which our knowledge is, at the best, vague and unsatisfactory. All that can be expected, therefore, in this respect, is that conditions shall not be required by a theory of morbid nutrition which are forbidden by the knowledge we have of normal nutrition. I believe it will be found that I have studiously kept within these limits; but, as I have already said, I am desirous that my views shall be examined by those who possess more profound physiological and chemical attainments than myself, in order that their truth may be strictly tested.

It is not my intention, then, to enter into a discussion of the purely physiological and chemical details of this hypothesis, but rather to direct attention to some of the most striking points in which it is consistent with the clinical history and pathology of tuberculosis, and especially to those in which it reconciles and explains apparently incongruous phenomena.

In the first place, it is necessary to impress the importance of the position occupied by fat in the processes of life; and I cannot do this better than by quoting the following statements by recognised physiologists, in which my own opinions are embodied.

"In considering fat as an important agent in the various phases of the metamorphosis of animal matter, we cannot, however, refer its action solely to mere contact or catalytic force; but we are constrained to assume that it co-operates in the metamorphic action, and experiences metamorphoses, combinations, and decompositions." (Lehmann's *Chemistry*, vol. i., p. 269.)

Under certain circumstances and for a certain time, "animals can be maintained by a nourishment from which fat is entirely excluded"; and, when the increase of fat in their bodies is in excess of the substances soluble in ether taken as food, "the formation of fat is due to a process of life." (Letter from Professor Liebig to the author, April 3, 1853.)

"In all the forms in which albumen naturally occurs, it is combined with fatty matter." (Kirkes's *Physiology*, 3rd edition, p. 12.)

"The usual function of fat is unquestionably, like that of starch or of sugar, to keep up the heat of the

animal. . . . . We require the combustion of all the non-nitrogenous constituents of food to enable us to account for animal heat. . . . . But it is, nevertheless, a fact that fat is always present in healthy muscle; and it is desirable to consider its relation to muscular action. . . . . The experiments made by Bidder and Schmidt on starving cats, and by Bischof and Voit on a starving dog, throw light upon this subject . . . When Bischof and Voit supplied their starving dog with fat, the waste of the body, as evidenced by the lessened amount of urea excreted, was diminished, because the fat supported the respiration, which before had partially to depend on wasting tissues. The fat cast over them a protective influence, and limited their waste to the support of their own dynamic functions; and in this fact would seem to be the use of fat after it is stored up in the muscle. We allude to its chemical use; for its mechanical advantage in lessening friction and its possible histogenetic employment in the formation of cells are not under consideration. . . . A man in ordinary health and activity wastes daily 1750 grains of dry flesh or 7000 grains of fresh muscle, which would contain 150 grains of fat. . . . We refer to fat distributed in and inherent to healthy muscles, and not to masses of adipose tissue. . . . The chemical use of fat deposited

within muscle may be to protect it from the assaults of oxygen during repose. A muscle, when at rest, gives out carbonic acid, which is, no doubt, partly due to the oxidation of its effete particles, but also to the oxidation of fat. . . . . Tissues may, and do, evolve heat by transformation when required to do so." (Dr. Lyon Playfair, *The Food of Man in Relation to his Useful Work:* 1865.)

A part of the heat necessary to maintain a constantly elevated temperature in birds and animals, "depends upon the *direct* combination of certain elements of the food with the oxygen of the air by the combustive process. The quantity of carbonic acid that is generated directly from the elements of the food, seems to vary considerably in different animals and in different states of the same individual. . . . . In man and other animals which can sustain considerable variations of climate, and can adapt themselves to a great diversity of habits, the quantity of carbonic acid formed by the *direct* combination of the elements of the food with the oxygen of the air differs extremely under different circumstances. It will serve as the *complement* of that which is formed in other ways." (Carpenter's *Principles of Physiology*, 6th edition, p. 265.)

"The fatty matters of the blood are obviously

destined to furnish the contents of the adipose and nervous vesicles, whilst their presence seems also to be required in the early stages of the production of cells generally. One of the principal sources of their expenditure, however, is that combustive process by which the heat of the body is maintained; and the amount deposited in the tissues as fat may be looked upon as the surplus of the quantity ingested that is not thus consumed. . . . The question of the capability of animals to produce fat has been much disputed. There seems, however, no reason for doubting that it may be generated, not only from the hydrocarbonaceous constituents of the food (sugar and starch), but also from the splitting up of the albuminous compounds." (Carpenter, *op. cit.*, page 341.)

"It is clear that fat exerts a protective influence over the albuminous tissues, sparing their consumption or oxidation by its own greater affinity for oxygen. . . . When fat is added to the food, its direct combustion takes up the oxygen, and prevents its action on the nitrogenous tissues." (Carpenter, *op. cit.*, p. 332.)

In the second place, I must point out that, although reliable observations on the blood in phthisis are very scarce, those which we have are

consistent with my hypothesis. "In this disease, the saponified fatty matters of the blood decrease perhaps more than in any other." (Becquerel and Rodier's *Animal Chemistry*, Speer's translation, 1857.)

In the next place, all reliable analyses and observations of the nature of tubercle itself give results consistent with my statement, that it is albuminoid tissue disintegrated by the combustion of some of its fat elements.

From the analyses of Scherer, "tubercle may be regarded as protein from which five atoms of carbon, one of hydrogen, and one of oxygen, have been removed." (Simon's *Chemistry*, translated by Dr. Day, p. 479.)

"Chemistry, equally with the microscope, has failed to point out any essential difference between 'grey miliary,' and 'crude yellow' tubercle. Chemists are agreed in regarding tubercle as consisting of some modified protein compound or compounds with fat and earthy salts." (Dr. Fuller, *Diseases of the Chest*, &c. p. 355.)

"It exhibits no tendency to form perfect cells, but rather abortive corpuscles, which form slowly, and slowly break down. Elementary molecules are not formed capable of further development. . . . . .

The tissue which is occasionally seen mixed with it in expectorated matter, is disintegrated lung (Andrew Clark), and only means that so much pulmonary structure has been destroyed." (Dr. Pollock, *Prognosis in Consumption*, p. 111.)

"The destructive change which takes place in tubercle is not a vital act, but is determined by a conversion of the chemical components arising out of an interchange of the elements." (Rokitansky, Sydenham Society's edition, vol. i., p. 299.)

In the fourth place, it must be observed that not only all the symptoms of confirmed phthisis, but the earliest appreciable symptoms of impending tuberculisation, upon which physicians are generally agreed, are entirely consistent with my hypothesis. These latter have been briefly and pointedly stated by Dr. Pollock in his recent work. "A pre-existing disorder of health is invariably present before there is any evidence of deposit in the lungs. . . . . These are symptoms not referable to any disease of the lung, so far as the function of respiration is concerned, but clearly indicative of a general disorder of the system. . . . . The earliest of all this group is emaciation." (*Op. cit.*, p. 39.)

The next point to which I would direct attention is the occurrence of hæmoptysis in the early stage of

phthisis. It so often happens that the first occurrence of hæmoptysis is followed by a steady descent of the patient, that, *popularly*, the hæmoptysis is looked upon as the causo of consumption. Although this is proved to be erroneous, it is a fact that, in a large proportion of cases, the first attack of hæmoptysis, even though slight in amount, is followed by marked symptoms of phthisis, and especially by loss of weight. The first Medical Report of the Brompton Hospital (1849) shows that hæmoptysis occurs in 63 per cent. of all cases of phthisis, and is more frequent in the first stage, as 3 to 1. Dr. Pollock says: "I have observed 351 cases of profuse hæmoptysis, of which 204, or more than one-half, occurred in the first three months of the illness." "An early profuse hæmoptysis is to be regarded as a bad prognostic; the softening being likely to take place early, and the remission in the progress of the disease to occur only at a period when already much structural mischief has taken place." "The prognostics to be derived from slight repeated hæmoptysis are, on the other hand, not favourable, this being the commencement of the ordinary form of phthisis." (*Op. cit.*, p. 139.)

This is entirely in accord with my hypothesis. For, so long as there is a sufficient supply of fat

in the system to protect the albuminoid tissues, there is no reason why tissue should be invaded; but, as soon as this ceases to be the case, the first breaking up of organic structure will occur; and this is the moment at which hæmoptysis may be reasonably expected to take place for the first time. The first hæmoptysis, therefore, although in no sense the cause of the tubercle or of the emaciation, may, in truth, often mark the point in the case at which the formation of tubercle begins, and also the point from which emaciation and other general symptoms of disease must advance in a marked degree until the progress of tuberculosis is stopped.

A sixth and still more interesting point is the relation of tuberculosis to the menstrual function, with respect to the effect upon the carbon in the system. Menstruation in health has a definite relation to the quantity of carbonic acid discharged from the system by other means. From the occurrence of puberty to the cessation of menstruation, so long as healthy nutrition is maintained, everything which arrests menstruation increases the discharge of carbonic acid from the lungs; and the recurrence of menstruation reduces that discharge. It is evident, therefore, that the menstrual flux is a means of eliminating carbon.

Now, it is well known that, previous to the appearance of local symptoms of tubercle, the catamenia usually become irregular, and gradually diminish in quantity until, as the disease goes on, they entirely cease; that permanent arrest of menstruation is a usual occurrence in confirmed and progressing tuberculosis; that any prolonged arrest in the disease is usually attended with a restoration of menstruation; that, when menstruation coincides with confirmed and progressing tuberculosis, all the symptoms of the disease are usually increased at the monthly period; that the cessation of menstruation during pregnancy is commonly accompanied by quiescence in the symptoms of tuberculosis; that a large number of cases of phthisis commence at the age of puberty; and (which appears at first very curious) that at this age, among those attacked, there is a large preponderance of males over females—("123 to 90,"—Pollock, p. 295).

As I read these facts, they have the following meaning. In females, the balance of their nutritive functions is so arranged that, at the age of puberty and during the whole generative period, there shall be, normally, a surplus of carbon in the system. At the age of puberty, this superfluous carbon protects them from some of the dangers of tuberculosis to

which boys, who have no such surplus to fall back upon, are exposed. Thus, a girl at the age of puberty, deprived of some of the normal supply of properly prepared fats by a temporary arrest of the function of the pancreas, simply suffers a delay in the appearance of menstruation, appropriating her surplus carbon for the protection of the tissues; whereas a boy, similarly placed, has no such means of escape.

The disappearance of the catamenia when tuberculosis has set in is explained by the deficient supply of fat to the blood cutting off the surplusage of carbon for excretion. The latency of tuberculosis, when the catamenia are retained by pregnancy, is explained by the saving thus temporarily effected in the expenditure of carbon. The restoration of the catamenia during periods of arrested tuberculosis is the expression of a return to the normal proportion of carbon in the system. With this clue, many other analogous phenomena may be easily explained. It is important to remember that, although the number of reliable experiments on the quantities of carbonic acid exhaled by the lungs in tuberculosis, as compared with the quantity in health, is not large; and that such experiments are open to many sources of fallacy; yet so far as they go they are to the effect

that the exhalation of carbonic acid from the lungs is diminished in tuberculosis, which is, like the above facts, in accordance with my hypothesis. (*Annalen der Chemie und Pharmacie*, 1846, p. 23.)

In the next place, I must point out that the order in which primary tuberculisation usually takes place in the principal organs, is consistent with my hypothesis and is explained by it.

The order is as follows. In adults and children, the lungs are by far the most frequent seat of tubercle. In adults, the small intestines and mesenteric glands are the most frequent seat of tubercle except the lungs. In children, the bronchial glands are affected with tubercle more frequently than any other part except the lungs, and they may be tuberculous without the lungs being so. After the bronchial glands, the small intestines and mesenteric glands are the most frequent seat, as in adults.

The lungs are the organs, in adult life, in which, *par excellence*, the required combination of conditions for precedence in tuberculisation exists. The small intestine and mesenteric system are the parts most nearly resembling the lungs in this respect.

It is hardly necessary to point out that there is no other part of the body traversed by fluids essentially the same as those which traverse the lacteals, mesen-

teric glands, thoracic duct, and pulmonary artery; and that there are no other parts of the body at which the relations between fats acted upon by the pancreas, albuminoid tissue, and oxygen, are so similar as in the small intestine and mesenteric system, and in the lungs.

In the lungs, the oxygen is in the air, and in the nutrient capillaries; in the intestine and mesenteric system, it is in the blood of the capillaries.

The explanation of the phenomenon, that the lungs occasionally give place to the bronchial glands and to the small intestine and mesenteric system in the precedence of tuberculisation, and that this occurs especially in children, is not easy. But the difficulty is partly due to a want of precise knowledge of the facts of the case. For example:

1. It is not certain, in the case of glands, that the tubercle is formed in them, not carried to them.

2. It is not certain how the interchange of oxygen from the air in the air-cells, with the carbonic acid in the blood of the pulmonary artery, takes place.

3. It is not certain to what extent the lung-tissue is nourished by the blood in the pulmonary capillaries.

4. It is not certain how the changes which take place in the fat that has been acted upon by the pan-

creas are effected during its course from the intestine to the pulmonary capillarics.

5. It is not certain what is the limit and essential condition of absorption of fat by the portal system.

So far, however, as these and other difficulties permit, my hypothesis offers a fair explanation of the phenomena of precedence in tuberculisation. The temporary protection to the lungs is afforded by fluid fats absorbed by the portal system, and thus carried to the lungs independent of the pancreas and mesenteric system. That the blood of the portal vessels is susceptible of such a function cannot, I think, be doubted, when we reflect that, in a normal state, the portal blood is twice as rich in fat as other venous blood; that, most probably, it is fluid oleinous fat, which is especially susceptible of absorption by blood-vessels; that the diet of children is peculiarly rich in such fats; and that in milk and cream the fats are minutely divided, and so arranged that in gastric digestion they must be mixed intimately with other foods, and thus be placed in the most favourable position for absorption; and when we also remember the readiness with which correlated organs assume compensatory functions.

It is, then, probable that, under certain circumstances, and especially in children, the olein carried

to the pulmonary circulation by the portal system may protect the lungs for a sufficient time to give other organs not so protected the precedence in the order of tuberculisation.

It is easier to see why, by these means, the precedence should fall upon the small intestine and mesenteric system, than why it should fall upon the bronchial glands. But it is possible that the protection given to the lungs may, in some cases, be sufficient to prevent disintegration of the albuminoid tissue during the *direct* combustive process, and thus to prevent *a primary deposit at the point of origin;* but not sufficient to prevent tuberculisation during the interstitial nutrition of the lungs; and thus the bronchial glands may suffer either because of the activity of their life in childhood, or because the disintegrated albuminoid matter is carried to them by the lymphatics as it is disengaged in the neighbouring tissues, constituting *a primary deposit distant from the point of origin.*

Supposing this explanation to stand, it involves also an explanation of the curious and inconstant relationship between fatty liver and phthisis; for which I have not been able to find any other that is more satisfactory.

In the years 1851-1853, I very closely and carefully investigated the question of fatty liver in

phthisis; examining and analysing a large number of cases and pathological reports, at St. Bartholomew's Hospital and elsewhere, in the hope of finding some explanation in the clinical histories of the cases. On referring to my notes of that date, I find the following summary. In cases of phthisis: 1. Fatty liver is not more frequent in those cases in which the normal structure of the lungs is extensively destroyed than in those in which such destruction is slight. It is not, therefore, due to diminished pulmonary function. 2. There is no constant relation between the condition of the appetite and the existence of fatty liver, nor between the degree of emaciation and fatty liver. 3. There is no constant relation between the state of the bowels or skin in fatty liver; they may both or one act in excess or not in excess, and fatty liver exist or not exist. (Diarrhœa is, however, more frequent in phthisis with fatty liver than without it.) These conditions may, one or all, be combined with great destruction of lung-substance and great emaciation and fatty liver exist or not exist. 4. The intimate relation between the portal and hepatic capillaries may allow the liver to become the seat of deposits from the elements of portal blood. 5. Supposing the portal system, in some cases of phthisis, to take on a compensatory function, and to absorb an inordinate quantity of oil

from the food, this might be deposited in the liver even though the rest of the body were emaciated.

To this summary, I may now add, that Dr. Ormerod's recent experiments on fatty degeneration (*Review of the Present State of Cardiac Pathology.* By E. L. Ormerod, 1864), show that the fat of degeneration is olein, not margarin; and this renders more probable the explanation I have offered of the occasional occurrence of fatty liver in phthisis from the undue absorption of fluid fats by the portal system.

NOTE.—The same number of the 'Brit. Med. Journal' in which my paper was published contained the following paragraph:—

"FATTY LIVER IN CHILDREN.—In the *post mortem* examinations of 222 children affected with adipose infiltration or with fatty degeneration of the liver, Drs. Steiner and Neureutter found that in 131 the age was from one to four years. Among the pathological conditions in the course of which fatty liver appears, the most frequent is tuberculosis; and the fact that this state in children is most frequently manifested as disease of the lymphatic glands and not of the lungs, negatives the supposition that the excessive deposit of fatty matter in the liver in connection with tuberculosis is due to deficient oxidation of hydrocarbons. Drs. Steiner and Neureutter consider rather—and in this they agree to some extent with Frerichs—that the origin of fatty liver is to be sought rather in the change in the constitution of the blood induced by the tuberculous disease, and that the liver may be fatty from the commencement of the tuberculous process. Next in order to tuberculosis in connection with fatty liver, is enteritis; which is not, however, to be regarded always as a cause. It may be preceded by the fatty disease; or in many cases, the two diseases are very

It is so obvious that all the phenomena in the following group are at once explained by my hypothesis, that I need not do more than place them in juxtaposition.

a. The rapid and marked improvement which takes place in many cases of phthisis when substances rich in olein are first supplied by the stomach.

b. The want of permanence in this effect (except under peculiar combinations of circumstances, which

probably due to a common cause. The exanthemata may also be followed by fatty liver; and the connection between these is only to be found in the changes of the blood. Fatty liver is also observed in connection with diseases of the bones in children, such as tuberculous caries and rickets. It is an error to ascribe the condition of the liver to the use of cod-liver oil in such cases; inasmuch as it is met with in children who have never taken oil, in as advanced a state as in those who have used oil for a year. Cases also of bronchitis, pneumonia, pleuro-pneumonia, and heart-disease, sometimes occur in which fatty deposit in the liver is met with. Of the causes indirectly affecting the liver, diet holds a principal place; inasmuch as the children have either a diet very rich in fat, or (especially among the poor) one deficient in fat but rich in hydrocarbons: and the mischievous influence of this diet is increased by the deficient metamorphoses resulting from want of exercise and from impure air. The authors draw a distinction between adipose infiltration and true fatty degeneration; and observe that the former coudition appears to be more frequent in children than the latter, inasmuch as it was met with in 188 cases out of the 222 examined. They hence conclude, that in by far the greater number of cases, fatty liver is not to be regarded as a result of malnutrition of the hepatic cells, but as the result of causes acting from without.—*Jahrbuch für Kinderkrankheiten;* and *Wiener Medezin. Wochenschr.*, December 6, 1865."

include the causes of restoration of pancreatic function).

c. The beneficial effect of a renewed exhibition of olein after it has been for some time withdrawn, although it had ceased to benefit while previously continued.

d. The repetition of these phenomena, with less and less marked benefit, and coetaneously with a steady decline of the patient.

e. In a large number of cases, ultimate intolerance of the olein, which previously had been much desired.

The next group of phenomena explained by my hypothesis is very curious. While engaged, during the year 1853, in the investigation of fatty liver in phthisis, to which I have referred, I was struck with the fact that the following group of phenomena may be found at the same time in a case of tuberculosis, and I attempted to discover a satisfactory way of explaining their apparent incongruities.

a. A considerable proportion of the lungs deprived of decarbonising and oxidising function.

b. A fair appetite and the ingestion of a normal quantity of food.

c. A difficulty in maintaining equalised animal heat throughout the body.

d. A steadily progressing loss of carbon from the tissues.

e. Blood not surcharged with carbon.

f. No sickness, diarrhœa, or other flux.

g. Arrested menstruation in females.

The most satisfactory explanation appeared to be an assumption, that the fat-elements of the food did not find their way into the blood of the right heart in normal proportions and conditions. With the hope of finding support for such an explanation in a coincidence of disease of the pancreas and tuberculosis, I searched and analysed the pathological reports at St. Bartholomew's and elsewhere, in which the condition of all the organs was to be found noted. But in every case of uncomplicated phthisis in which the pancreas was mentioned, it was reported "healthy."

In consequence of this disappointing result, I set down as subjects for future work, the questions:

1. What evidence can be found that the pancreatic secretion can be deprived of its normal properties without leaving traces of structural disease in the pancreas?

2. Are there not affections of the pancreas competent to deprave its secretion, but which have not hitherto been recognised as disease?

Both of these questions may now be answered in the affirmative, almost with certainty.

In answer to the first, I must refer to the careful and often repeated experiments of Professor Bernard. They show that when the nerves of the pancreas are acted upon, either by excitation of the cerebro-spinal or by section of the sympathetic fibres, the secretion grows abundant and uninterrupted, while a profuse diarrhœa is constantly established. Extirpation of the semilunar ganglion produces similar effects; and under these special conditions the pancreas pours forth a peculiar fluid which no longer exhibits the physiological properties of the secretion. When the secretion is thus rendered continuous, the characteristic active principles are no longer produced within the gland, and the watery vehicle alone escapes from the secreting apparatus. Bernard particularly insists upon the important fact that "the general perturbations of the economy" exert a powerful influence upon the functions of the pancreas, and the least degree of inflammation in its neighbourhood perverts the properties of the pancreatic juice. (Bernard's Lectures on Physiology, *Medical Times and Gazette*, 1860.)

I have been informed by a butcher who deals largely in calves' sweetbreads, and is a shrewd and

observant man, that there is a great difference in the qualities of sweetbreads, dependent upon the previous state of the nervous system of the animal. If the calf has been brought to London comfortably in a cart, the sweetbread is generally in good condition; but, if it has been "worried about," he is often obliged to throw the sweetbread away, as not good enough for sale.

From the effect of nervous influences upon secretion generally, especially upon that of the salivary glands, the stomach, and the mammary gland (see Carpenter, 6th Edition, pp. 738 to 744), we might reasonably expect by analogy that the pancreas would suffer in the manner shown by Bernard.

It is evident, then, that the pancreatic secretion can be deprived of its normal properties by causes acting through the nervous system, both cerebro-spinal and sympathetic; and by affections of neighbouring organs. Such causes may be of a temporary or of a more permanent nature, and, unless of long duration, may not leave traces of structural disease in the pancreas.

I particularly desired that, before publishing my hypothesis, I might be able to bring forward absolute proofs of the nature and extent of the perverted or arrested function of the pancreas in tuberculosis,

in the form of results of direct experiments upon the pancreatic juice of persons dying of phthisis. Through the kindness of Drs. Quain and Symes Thompson at Brompton Hospital, of Dr. Cayley at the Middlesex, and of Dr. Andrew at St. Bartholomew's, together with my own opportunities at the Royal Infirmary, I was able to obtain a supply of specimens; and, with the invaluable chemical assistance of Mr. Heathorn, I commenced a systematic examination of the properties of the pancreatic secretion in phthisis. These exceedingly troublesome experiments were carried on with great care from the beginning of November 1864 till the beginning of March 1865, and a detailed report was kept of the results; but I regret to say that they were obliged to be finally abandoned, from a conviction that we were wasting our time. The results were valueless, for the following reasons:

1. We had no standard of healthy pancreatic secretion of comparison.

2. Our attempts to form such a standard were frustrated by the impossibility of getting the human pancreas under circumstances which did not vary considerably in the following essential conditions:

a. Number of hours between death and the last meal.

b. Character of the last meal.

c. Cause and mode of death and nature of co-existent diseases.

d. Number of hours between death and the *post mortem* examination.

e. Number of hours between death and the commencement of experiments on the secretion.

These obstacles to the establishment of a healthy standard all equally existed in the case of diseased specimens. In addition it was proved by experiments upon the lower animals, that no reliance can be placed upon the characters of the pancreatic secretion when not examined till twelve hours after death; whereas the average number of hours between death and *post mortem* examination for all of our cases was 27·5 hours, the minimum being 9, the maximum 54. I give these details of failure in the hope that some one working within the walls of a hospital may find it practicable to get over these difficulties, and to prosecute this important inquiry under conditions which justify confidence in the results. To any one undertaking such experiments, I shall gladly give any assistance in my power. I can at least point out some sources of fallacy to be avoided, and some of the means of success.

In answer to the second question—"Are there

not affections of the pancreas competent to deprave its secretion, which have not hitherto been recognised as disease ?"—I am permitted by Dr. Fenwick to state in general terms, that the results of a large number of examinations made by him of the pancreas in man and the lower animals, show a peculiar liability of this organ to different forms of degeneration. But as it is probable that Dr. Fenwick will himself make public his observations, I forbear from giving them in more detail.

The fact, then, which in 1853 appeared to show that defective pancreatic action could not be essentially connected with tuberculosis—viz., that, in all the cases of uncomplicated phthisis that I could discover, in which the pancreas had been examined, it was reported healthy—need no longer be considered of any value as an argument against my hypothesis; whereas, on the other hand, the observations of Professor Bernard must be considered as important evidence of its truth.

But the most remarkable evidence of the truth of my hypothesis is this, that it explains in the simplest manner a mass of apparently incongruous facts in the etiology of tuberculosis, which no explanation hitherto attempted has been at all competent to deal with in a group. I refer to

such established and recognised causes of phthisis as are agreed upon by the general verdict of physicians and the public. I will abstain from adding any that I might myself be disposed to suggest, and only select from reliable authors those which are apparently *least* in accord with each other.

The question is, How can a constant result—viz., the formation of nascent tubercle—be produced by the following causes?

1. Disappointment; home-sickness; longings after the objects of affection, either absent or lost; and other depressing influences.

2. Whatever depresses vital power—exhausting passions, poverty, and the vicissitudes of life.

3. Persistent defective expansion of the chest, and defective exercise of the respiratory powers.

4. Persistence of young persons in a diet deficient in milk.

5. Inattention to a due preservation of the cutaneous function.

6. Chronic alcoholism.

7. Neglect of exercise in the open air; deprivation of sunlight; congregation of numbers in a close or insufficiently ventilated place. "If disease of a given form be so associated with certain conditions that, in two-thirds of the instances which

present themselves to our notice, these conditions be present, we cannot avoid referring the diseased result to the antecedent error. In other words, deficient ventilation and crowded apartments are eminently productive of tubercular disease. . . .The effects of posture are chiefly observable in bootmakers, tailors, hand-weavers, and others whose work necessitates the stooping posture. The results are mechanical hindrance to the free entrance of air to the chest, restricted expansion of the body wall, and an imperfect respiration. In the case of bootmakers, there is the addition of pressure on the epigastrium, which gives rise to the well-known neurosis. But, on the whole, all the errors of this class are to be referred to insufficient expansion of the chest." (Pollock, *Elements of Prognosis in Consumption*, p. 368.)

8. " Rebreathed air."
9. Cyanosis.
10. Diabetes.
11. Acute diseases, especially continued fever.
12. Childbirth.
13. Superlactation in mothers.
14. Weaning in children.
15. Hereditary taint.

All these causes of phthisis may be assembled under four heads.

1. Causes which act directly upon the pancreas, either especially, or in common with other internal organs; *e.g.*, hereditary tendency to disease of the pancreas; occupations causing pressure upon the abdomen in the pancreatic region; acute diseases, especially continued fevers; inflammatory affections of neighbouring parts.

2. Causes which act indirectly upon the pancreas, by diminishing the elimination of carbon from the blood, and thereby reducing the normal call for the introduction of fat from the food into the blood. By these means, the pancreas is kept in a state of inactivity and low nutrition, until in time its secreting powers are depraved or destroyed, and the organ itself becomes degenerated, in accordance with the laws governing all other secreting organs.

It is evident that this heading will include a large number of the causes of phthisis which I have enumerated; *e.g.*, all those which diminish respiratory blood-changes for protracted periods, whether it be simply through deficient expansion of the chest, or through the hypercarbonetted condition of the air presented for respiration; and all causes of deficient excretion by the skin, especially if combined with defective respiratory action. It will also include cyanosis and chronic alcoholism.

Cyanosis is a condition which would, at first

thought, appear to be antagonistic to my hypothesis: for, if tubercle is produced by the combustion of the fat elements of the albuminoid tissues, it would seem that a disease in which the blood is persistently surcharged with carbon would, *par excellence*, be protective against tuberculosis. But the contrary is the fact; and it is easily explained. It is only protracted cyanosis that ends in tuberculosis. In these cases the long continuance of hypercarbonetted blood paralyses the function of the pancreas. But, as it is not carbon only that is requisite to maintain nutrition and protect against tuberculosis, but fat properly prepared by the pancreas,—after a time the albuminoid tissues are invaded to supply the fat-elements which the pancreas has lost the power to prepare, and tuberculisation results.

In chronic alcoholism, the mode of operation is somewhat similar to that in cyanosis. A form of hydrocarbon is thrown into the circulation through the portal system, which substitutes the normal supply of fats by the lacteal system. The affinity of oxygen for alcohol being greater than its affinity for fat, respiration is supplied from this artificial source with carbon for direct combustion; an artificial nutrition is kept up, in which the

natural call for fat is stopped, and the function of the pancreas is reduced to supplying the minimum quantity necessary for histogenetic purposes. To this inactivity of the organ is added the usual tendency to degeneration due to alcoholism. In course of time, the pancreas loses that minimum amount of function which it had been allowed to exert, and fails to supply even so much fat as was necessary to protect the albuminoid tissues, and tuberculosis results. Or, as frequently happens, the toper ceases to obtain his supply of alcohol, either from inability to get it, or from inability to absorb it or to retain it on the stomach. His artificial supply of carbon, upon which he has been depending, is thus cut off; a sudden call is made upon the pancreas for that which it has now lost the power to give; the tissues are disintegrated to supply the required fat-elements; and tubercle is produced.

3. Under a third head may be included causes which deprive the system of carbon to such an undue extent that the pancreas cannot keep pace with the demand made upon its function; the supply of fat to the blood becomes insufficient to keep up the waste; and the albuminoid tissues are invaded. This may be the case when those who inherit a

tendency to disease of the pancreas, or who have acquired a feeble secreting power in the organ, are exposed to drains upon their fat-elements which in healthy persons might be met by increased function. It is thus that, in tuberculous families, childbirth, and superlactation, especially when not protected by proper diet and regimen, precipitate the patient into tuberculosis; and, as the mother is deprived of fat-elements by lactation, so is the child deprived of them by a persistence in a diet deficient in milk. In the case of the child thus deprived of fat, a double injury is done—first, by cutting off the supply of fat-elements necessary for the protection of the tissues; and, secondly, by paralysing the function of the pancreas by prolonged inactivity.

Under this heading also must be placed Diabetes. Tuberculosis occurs in diabetes when the excessive drain upon the carbon by the excretion of the carbo-hydrates, as sugar, is not duly supplemented by a corresponding supply of fat in the food; or when the demand for pancreatic action has become so excessive that the pancreas is no longer able to keep up to it. Then the albuminoid tissues are attacked, and tuberculisation occurs.

4. A fourth heading will include all causes which act powerfully in depressing the nervous system,

either suddenly or gradually, and by this means pervert or paralyse the secreting function of the pancreas. It is in this way that "the vicissitudes of life" bring on tuberculosis, especially in those who by hereditary transmission have a tendency to depraved pancreatic function, or in whom the causes of nervous depression are conjoined with some of the other causes of disease of the pancreas which have been enumerated. Under this fourth heading, therefore, we must place all forms of shock to the cerebro-spinal or ganglionic nervous systems, and all such causes of mental and nervous depression, as disappointed love or ambition, excessive grief, unrequited longings, hope deferred, and the like.

Having given a fair example of the way in which my hypothesis coordinates and explains apparently incongruous and irreconcilable phenomena connected with tuberculosis, let me point out that it also affords a satisfactory explanation of the variable course of tuberculosis in different cases.

1. A sudden, almost complete, or total suspension of normal pancreatic secretion, accounts for acute tuberculosis.

2. A less complete suspension or perversion of the function of the pancreas accounts for chronic tuberculosis.

3. Either of the above changes of pancreatic secretion, occurring intermittently, accounts for recurrent tuberculosis.

I have next to speak of that portion of the hypothesis which assumes a change in the absorbing power of the small intestine, by which the passage of fats into the blood may be prevented after the function of the pancreas has been restored.

That the continual passage, through the delicate absorbent apparatus of the intestinal mucous membrane, of a depraved pancreatic secretion, and of food that has not undergone those changes which fit it to be brought into contact with the absorbing surface, must damage the apparatus, is a conclusion which analogy justifies us in assuming to be correct. That such damage does occur in the alimentary canal of the tuberculous, is amply proved by pathological research; and the extreme difficulty of identifying, after death, delicate changes in the condition of mucous membranes, renders it certain that such changes may continually occur without being recognised in a *post mortem* examination.

Bernard found that, in dogs who had been deprived of their pancreas, the fæces commonly became streaked with blood, as from ulceration of the intestines. (*Mémoirs sur le Pancreas*, 1851.)

Bidder and Schmidt observed, in their experiments on the lower animals, that inflammation of the mucous membrane of the intestine at once prevented the absorption of fatty chyle by the lacteals.

To say the least, it is in the highest degree probable that perverted pancreatic secretion, and food improperly prepared for absorption, passing over the mucous membrane of the intestine from day to day, do, sooner or later, so damage its absorbing function as to add an additional obstruction to nutrition by the lacteal system; and, as Bernard has shown that the general perturbations of the economy, and the slightest inflammation in the neighbourhood of the pancreas, especially in the intestine, pervert the properties of the pancreatic juice, any damage done to the mucous membrane of the neighbouring intestine by imperfect pancreatic action must react upon its cause, and throw a double difficulty in the way of restoring either the pancreas or the mucous membrane to a healthy condition.

NOTE.—From my own observations, I am disposed to think, that laryngeal phthisis will be found to occur more frequently in persons who have slept, night after night, with patients suffering from the last stage of consumption, than in other persons; and that molecules of tubercle may, in this way, be occasionally transplanted by the breath from the lungs of one person to the

respiratory passages of another; then being carried by the lymphatics into the circulation, producing tuberculæmia and secondary deposits (see note p. 6). I am not prepared to say this is the case; but to suggest it as an important subject for further clinical and pathological observation.

Treatment.

Assuming my hypothesis to be correct in its chief parts, the following are the principles of treatment which it indicates.

1. If the case is so far advanced that it is considered hopeless to aim at restoring the function of the pancreas and the absorbing power of the intestine, two principles must guide our treatment.

A. To supply the greatest amount of fat to the blood that is possible by other means than through the action of the pancreas.

a. By giving pancreatic emulsions of solid fat, or if these be rejected, pancreatic emulsions of oil, in the hope that some absorbing power for such matters may yet remain in the digestive tract.

b. Chiefly, by giving olein, with a view to its absorption by the portal system of vessels: and by rubbing it into the surface of the body and limbs, with a view to its absorption by the skin.

c. By giving highly albuminous food in conditions in which it may be most easily utilised, so as to

supply an excess of material as a source of carbon by disintegration.

B. To save the albuminoid tissues from disintegration to the greatest possible extent.

a. By supplying a surplus of carbohydrates, so as to economise as much as possible the consumption of hydrocarbons.

b. By diminishing the proportion of oxygen in the air presented to the lungs.

c. By diminishing the demand for the generation of animal heat, by supplying it artificially.

d. By reducing respiratory and cutaneous action to the lowest point consistent with maintaining what remains of appetite and digestive power.

It is obvious that the persistence in these means must be considered utterly at variance with curative intentions, and simply as the expedients best fitted to prolong the process of gradual death.

2. If the symptoms are only what is commonly called "premonitory"—that is, if they are those of commencing tuberculosis, and no reason or sign is discoverable which justifies the suspicion that tuberculisation has commenced; if a sufficiency of fat-elements remains, without calling upon the albuminoid tissues—*the principles of treatment are quite opposite to those last detailed.* They are now entirely curative in their intention.

A. To restore the function of the pancreas as quickly as possible, by placing the patient under those conditions which call for pancreatic action.

a. An atmosphere rich in oxygen.

b. A climate at once cold and dry.

c. Exercise of the respiratory and cutaneous functions.

d. Cheerfulness of the mind and quiet to the emotions; exhilaration of the animal spirits.

e. A good mixed diet easily digested, and free from substances irritating to the mucous tract.

f. Pancreatic emulsions of solid fat, and pancreatic juice, in quantities sufficient to ensure protection to the albuminoid tissues while the process of restoration is going on in the pancreas, but *not in such quantity as to entirely supplant* the natural pancreatic function and thus to counteract the measures adopted for calling it into action.

These two classes of cases represent the two extremes in the history of tuberculosis—the greatest and the least hope of cure.

3. A third class, intermediate between these extreme points, includes the bulk of all cases brought under the care of the physician. They require a combination of the two principles of treatment already stated. The nice adjustment of these two

plans of treatment—the hopeful and the hopeless, the palliative and the curative—calls for all the intelligence and judgment that a medical man can possess.

If tuberculisation, even in the smallest degree, is going on, the first and most urgent need is to supply fats. Olein should be given for absorption by the portal system, thus to afford, so far as it is able, materials for combustion. But, above all, FATS ACTED UPON BY PANCREATIC JUICE are called for, and must be supplied until the deficiency is removed, the just balance restored, the process of tuberculisation stopped. Until this point is arrived at—until the balance is turned in favour of the albuminoid tissues —everything which favours the reception of oxygen into the blood, everything which increases the wear and tear of the body, everything which calls for the generation of animal heat, directly favours tuberculisation, and precipitates the patient into the very catastrophe we wish to avert.

On the other hand, as soon as the balance has been turned in favour of the albuminoid tissues, and tuberculisation has been artificially arrested, everything which postpones the restoration of the function of the pancreas directly favours the conversion of a temporary into a permanent disease. But

moderation is essential to success. Too great and too sudden a demand upon a weak and defective pancreas may only paralyse the organism in its attempts at restoration of function. It is, however, impossible to magnify the importance of periods of arrested tuberculisation, whether artificially or naturally produced. They are the bright opportunities for the permanent restoration of pancreatic function. Too often these opportunities are wasted. The patient and his friends, pleased with the marked improvement that has taken place under some plan of treatment, either continue this treatment long after its proper time and place have passed by, or give up treatment altogether, content to wait till signs of active disease return. In either case, an opportunity for establishing a permanent recovery is thrown away, and perhaps it may never return.

It must not be forgotten that, when tuberculisation and tuberculosis have been stopped, when the function of the pancreas has been restored and the intestinal mucous membrane brought back to a normal condition, there may yet remain *tuberculæmia*. From time to time, fresh blood-poisoning may occur from the absorption of deposited tubercle; and, if there be any considerable quantity deposited, this chronic blood-poisoning may become a tedious and

dangerous disease. The absorbed tubercle may be again deposited and again absorbed, and thus keep up a ceaseless repetition of morbid changes, somewhat analogous to those witnessed in pyæmia. Treatment directed to the blood-poisoning will, therefore, be imperatively called for in most cases of tuberculosis which have advanced to the stage of tuberculisation.

I have long been aiming at testing my views regarding the action of the pancreas in tuberculosis by a crucial experiment; but the difficulties in the way of artificially supplying pancreatic juice to a number of patients were so numerous that they were not finally overcome till the summer of 1863. With the valuable assistance of Mr. Heathorn, who had long been working with me at the subject, I succeeded in obtaining a pancreatic emulsion of fat sufficiently palatable and in sufficient quantity to permit of my beginning my crucial experiments by administering it to patients suffering from tuberculosis, at the Royal Infirmary for Diseases of the Chest. For some time our supply was too fluctuating to allow of the experiments being carried out in a manner that I thought justified me in making the results public; but in the summer of 1864, I was able to draw up the first of the three reports which have

since appeared in the *Lancet*. This was published September 10th, 1864; the emulsion used in the reported cases being made of beef-fat and the pancreatic juice of the pig. The next report was published June 10th, 1865; the emulsion used in these cases being made of lard-oil, and the pancreatic juice of the pig. The last report was published November 11th and 18th, 1865; some of the cases having been treated with the lard-oil emulsion, and some with an emulsion of suet and pancreatic juice of the pig. The number of cases in the three reports is eighty-nine; but, out of these, only twenty were in the first stage, and in these the deposit of tubercle was considerable, and the general symptoms very marked; in fact, they were, as a rule, just passing into the second stage, before applying for treatment at the hospital.

It is obvious, therefore, that, although the results were remarkably satisfactory, *there was not in one of the cases the opportunity of trying the treatment in the most hopeful stage*. In addition to this, I must remark, that for a long time, until I was satisfied of the good effects of the emulsion, I did not feel justified in giving it unless for some reason the patient refused to take cod-liver oil; so that this added another unfavourable feature to the cases

selected for experiment. And, again, it is important to bear in mind that the emulsion, as we first prepared it, was necessarily in a much less satisfactory condition than the emulsion used in later experiments. From time to time, Mr. Heathorn introduced great improvements in the mode of manufacture, and these have been still further carried out by Mr. Schweitzer; so that the emulsions, as now sold by Messrs. Savory and Moore, are far more satisfactory in every respect than those which we employed for the hospital cases included in my reports.

Those accustomed to hospital practice will know well that the chances of treating tuberculosis in its simple and early stage, before the occurrence of tuberculisation, are very rare. Patients do not apply at hospitals for relief until they have some marked symptoms of disease, such as hæmoptysis or purulent expectoration. When by chance they are seen in an earlier stage, they discontinue treatment directly the first feeling of debility is allayed. It is in private practice that the opportunity arises for detecting commencing tuberculosis; and it is especially within the province of the family doctor, frequenting the houses of his patients for other maladies, to keep a watch, especially in consumptive

families, for the earliest dawn of impending tuberculosis.

In conclusion, I wish particularly to impress that, if the views here brought forward are correct, the importance of pancreatic emulsions of solid fat over cod-liver oil, in the treatment of tuberculosis, must be as great as that of supplying a plant with good soil instead of putting it into water. In the one case, a provision is made for the maintenance of normal life; in the other, death is only postponed by a temporary expedient.

FIRST REPORT

OF

Cases Treated with Pancreatic Emulsion,

AT THE ROYAL INFIRMARY FOR DISEASES OF THE CHEST, CITY-ROAD.

[From the Lancet, September 10, 1864.]

A NUMBER of practical difficulties presented themselves, in carrying out this plan of treatment; many of which will be familiar to those who have experimented with pancreatic juice even on a small scale; but these were materially increased by the circumstance of having to keep up a constant supply to a number of patients, and by the patients being treated out of hospital, and, therefore requiring to take home with them the quantity of medicament necessary to last till their next visit to the infirmary. Many different experiments* were

* We regret to be unable to find space for the long and elaborate tables and notes appended to this paper, and containing the observations on which it is founded.—ED. LANCET.

made before a plan was fixed upon which appeared practicable; and I cannot too warmly praise the care, skill, and intelligence of Mr. Heathorn,—the scientific and practical chemist of 94, St. John's-street-road,—who assisted me in overcoming these difficulties, and afterwards prepared the pancreatic juice for administration at the infirmary.

The plan we finally decided to follow was, to prepare an emulsion of beef-fat with the pancreatic juice of the pig, of the consistence of thick Devonshire cream; to supply the patient with this emulsion in a covered jar holding a week's allowance, and to order each dose to be taken stirred in milk. The emulsion mixed with the milk without any difficulty, and in the proportion of half an ounce in a breakfast-cupful of milk, was not at all an unpleasant drink.

Comparative specimens were prepared of cod-liver oil and of beef-fat, emulsified with liquor potassæ and with pancreatic juice, and submitted to microscopical examination.

The following report was made by my friend Mr. Farrants, late President of the Microscopical Society, on specimens submitted to him in March, 1864.

Microscopical Examination of Emulsions of Cod-liver Oil and Beef Fat with Liq. Potassæ and with Pancreatic Juice.

DIAMETERS OF GLOBULES.

Cod-liver Oil and Liq. Potassæ:

α Largest, $\frac{1}{800}''$ ·0012″ .. Numerous.
β Commonest, $\frac{1}{3000}''$ ·0003″ .. Very common.
γ Smallest, $\frac{1}{10000}''$ ·0001″ .. As numerous as preceding.

Emulsion thin, readily separating into two layers.

Cod-liver Oil and Pancreatic Juice:

α Largest, $\frac{1}{300}''$ ·0030″ .. In considerable number.
β Commonest, $\frac{1}{1250}''$ ·0008″ .. Common.
γ Smallest, $\frac{1}{16000}''$ ·00006″ .. Abundant.

In this emulsion larger globules occur, and are more common than in the first. The smallest met with are smaller and in much greater number than in the potash emulsion. The oil is more minutely divided; the emulsion is thicker and more permanent.

Beef Fat and Liq. Potassæ:

α Largest, $\frac{1}{1600}''$ ·0006″
β Commonest, $\frac{1}{2500}''$ ·0004″
γ Smallest, $\frac{1}{10000}''$ ·0001″

But few clearly-defined globules distinguishable. The fat is almost entirely saponified.

Beef Fat and Pancreatic Juice:

α Largest, $\frac{1}{1400}''$ ·0007″ .. Not numerous.
β Commonest, $\frac{1}{2500}''$ ·0004″ .. Far most common.
γ Smallest, $\frac{1}{10000}''$ ·0001″ .. Uncommon.

Emulsion fairly uniform. No tendency to separate.

The liq. potassæ emulsions differed greatly from those with pancreatic juice, the latter being much more complete and permanent emulsions, the former being more or less complete soaps. The smallest globules seen were those of cod-liver oil and pancreatic juice—viz., $\frac{1}{10000}$ of an inch, which were abundant. This would probably have been the best for my experiments; but I selected the beef-fat emulsion in preference, in order that there should be no confusion between the ordinary effects of cod-liver oil and the effects of the pancreatic juice.

The beef-fat emulsion remained complete after standing two days, mixed with pepsine, hydrochloric acid, and water.

As I do not propose on this occasion to enter into a discussion of the facts produced, but simply to submit them to the consideration of others, it will not be necessary to make any further introductory remarks, except to mention, in explanation of some things that may be noticed in the following analyses of the cases—1st, that the experiments were made upon out-patients; 2nd, that an out-patient's letter at the Royal Infirmary only lasts eight weeks; 3rd, that as a rule the emulsion was not ordered unless the patient objected to cod-liver oil. Those who have attempted to keep notes of cases amongst out-patients

know how difficult it is to do so satisfactorily; how much trouble is often thrown away, by patients, who think they are getting well, discharging themselves without giving the physician the opportunity of making the final report, necessary to show the progress of the case. It often happened so in my cases, and they had to be discarded from the tables, although it will be seen that I have retained a few in which all the points were filled up except the last. Some cases had to be rejected after they were completed, in consequence of facts turning up in their course which made it doubtful whether the original diagnosis of consumption was unquestionably correct. With these exceptions, no selection was exercised; the cases were taken for experiment in the order in which they applied for admission, and the particulars registered under the following principal headings:—1, Name; 2, occupation; 3, age; 4, sex; 5, condition on admission (*a*, general symptoms; *b*, physical signs); 6, condition on discharge (*a*, general symptoms; *b*, physical signs); 7, quantity of emulsion taken per day; 8, how long continued; 9, effects; 10, effects of cod-liver oil; 11, other treatment; 12, notes.

The results of treatment in 33 cases retained for analysis may be seen in the following;—

Summary of Thirty-three Cases of Consumption treated with Pancreatic Emulsion of Beef Fat :—

7 cases in the 1st stage—Condition on discharge measured by the general symptoms: Improved, 7; stationary, 0; worse, 0; no final report, 0.

Condition on discharge measured by physical signs; Improved, 0; stationary, 7; worse, 0; no final report, 0.

Emulsion agreed, 6; emulsion disagreed, 1.

Cod-liver oil agreed, 3; disagreed, 1; not tried, 3.

14 cases in the 2nd stage—Condition on discharge measured by the general symptoms: Improved, 9; stationary, 2; worse, 1; no final report, 2.

Condition on discharge measured by physical signs: Improved, 8; stationary, 2; worse, 2; no final report, 2.

Emulsion agreed, 14; emulsion disagreed, 0.

Cod-liver oil agreed, 5; disagreed, 5; not tried, 4.

12 cases in the third stage—Condition on discharge measured by the general symptoms: Improved, 8; stationary, 0: worse, 3; no final report, 1.

Condition on discharge measured by physical signs; Improved, 5; stationary, 1; worse, 3; no final report, 3.

Emulsion agreed, 10; emulsion disagreed, 2.

Cod-liver oil agreed, 5; disagreed, 5; not tried, 2.

The whole 33 cases measured by general symptoms: Improved, 24; stationary, 2; worse, 4; not noted, 3.

The whole 33 cases measured by physical signs: Improved, 13; stationary, 10; worse, 5; not noted, 5.

Emulsion agreed, 30; disagreed, 3.

Cod-liver oil agrced, 13; disagreed, 11; not tried, 9.

Average quantity of emulsion taken by each patient about 1 oz. avoirdupois in 1 pint of milk each day for eight weeks.

A SECOND REPORT
OF
Cases Treated with Pancreatic Emulsion,
AT THE ROYAL INFIRMARY FOR DISEASES OF THE CHEST, CITY-ROAD.

[From the Lancet, June 10, 1865.]

SINCE my last report on this subject in THE LANCET of September 10th, 1864, a considerable number of cases have been placed under treatment with the pancreatic emulsion of lard-oil described in my letter of October last.* This emulsion has proved more convenient for use and more uniform in quality than the beef-fat emulsion used in the first set of cases, and the results are very satisfactory. Nevertheless, I am still of opinion that it may be important to administer solid fats rather than oils; and, as Mr. Heathorn has succeeded in preparing a very fine and uniform emulsion of suet, some comparative experiments with this are now in progress, the results of which shall be reported when complete.

* See Lancet, October 1, 1864.

Many of the cases under the lard-oil emulsion are still under treatment, and must be reserved for a future report; but I am able to give a summary of sixteen which have already been discharged. I regret that the details of the cases are too long for the columns of THE LANCET, but I shall be happy to show them to any medical men who are interested in the subject. It is necessary to mention, in explanation of the fact that cod-liver oil disagreed with so large a proportion of these patients, that, as a rule, the emulsion was not given unless there was some reason for discontinuing cod-liver oil. It will be seen from this that the cases treated with emulsion were of a class unfavourable for success.

It should also be borne in mind that cases of consumption do not present themselves for treatment at hospitals in the *first* stage of the disease, unless the symptoms are very marked; and therefore the class of cases most favourable for success are virtually excluded from hospital practice. The earliest stage of tuberculosis, before the vital powers have been seriously reduced or a large amount of fat has been removed from the tissues, is that which should be selected for curative treatment.

The effects of the emulsion on the digestive

functions may be partly learned from the following short extracts from the cases.

EFFECTS OF COD-LIVER OIL AND OF EMULSION.

FOUR CASES IN THE FIRST STAGE.

Case 34.—Has tried oil, and it "won't keep down." Emulsion agrees well; and at the fourth week he says, "I never had anything to do me so much good in my life." Appetite good while taking it.

Case 42.—Has taken oil one month with some improvement; it does not destroy appetite, but it rises, and is very offensive. Emulsion agrees; she likes it much better than oil. After taking it a month she could not be persuaded to try oil again. Does not rise, or destroy appetite.

Case 45.—Has taken oil three months, with slight increase in flesh, and it agrees; but chest symptoms are getting worse. Emulsion agrees well; he says "it's very nice." After fourteen days, very much improved in all respects.

Case 49.—Has been taking oil and tonics, but loses flesh and strength, and gets rapidly worse; appetite is destroyed while taking oil, but she is so convinced of the importance of taking it that

it is difficult to persuade her to take emulsion instead. Emulsion agrees. On third week, she says she "gets on wonderfully:" and on fourth, that she "eats till she is ashamed of herself." On fourth week, excessive craving gone; appetite regular and good; gaining strength rapidly.

SIX CASES IN THE SECOND STAGE.

Case 38.—Oil produces constant nausea, but does not rise. Emulsion agrees well; does not leave nausea, like the oil. At fourth week, oil tried again, but it still produces nausea; obliged to return to emulsion.

Case 43.—Oil produces nausea, and, after a week or two, cannot be kept down—has often been tried; can't take any sort of fat. Emulsion agrees well; appetite improves while taking it, and the frequent bilious feelings to which he was subject have disappeared. At fourth week, enjoys fat; and can now take cod-liver oil, as well as emulsion, without nausea.

Case 44.—Cannot be induced to take oil, dislikes it so much. Emulsion agrees; never rises, and is taken with ease. At third week, says flesh gets firmer.

Case 46.—Cannot take oil; has often tried it; it

produces nausea and biliousness. Emulsion agrees; appetite improves while taking it.

Case 48.—Oil agreed formerly, and did good, but for the last twelve months it has disagreed more and more, and for some time past will not keep down at all. Emulsion agrees, no nausea produced. At fourth week, appetite is improved, especially for meat; says he "feels quite a different man."

Case 53.—Has taken oil six months, but it spoils appetite, and he loses flesh and strength. Emulsion agrees: he likes it. At fourth week, appetite is very good; is able to take suet with pleasure, and says he could take more emulsion if allowed.

SIX CASES IN THE THIRD STAGE.

Case 36.—Cod oil has often been tried, but it always produces bilious sickness after about a week; loses flesh fast. Emulsion agrees well; appetite improves; no nausea or bilious feeling. After ten weeks, says she is getting "tired of emulsion," but it still agrees. As she can eat fat with pleasure, ordered suet and milk instead of emulsion.

Case 39.—Four years ago took cod-liver oil with advantage, but of late it rises, and brings up the food with it. The first two doses of emulsion rose, but it afterwards agreed, and is now liked.

At fourth week, cod oil tried again, but "won't keep down." Emulsion resumed, and agrees; appetite good; enjoys meat while taking emulsion.

CASE 40.—Cod oil agreed last winter, but now it "won't keep down." Emulsion agrees well. At fourth week oil tried again, but still "won't keep down." Emulsion resumed, and taken six weeks with pleasure.

CASE 47.—Cod oil can be taken, but it produces nausea, and appetite is very bad while taking it. Emulsion agrees, does not produce nausea, and appetite steadily improves while taking it.

CASE 50.—Has tried cod oil, but "can't take it." Emulsion agrees well; appetite for meat improved.

CASE 54.—Oil tried two months ago and also this week, but he brings up his food while taking oil, and loses ground fast. Emulsion agrees; gains flesh and strength. After taking emulsion five weeks, can take two ounces of oil and emulsion as well.

Summary of Sixteen Cases of Consumption treated with Pancreatic Emulsion or Lard Oil.

4 cases in the 1st stage — Condition on discharge measured by general symptoms: Improved, 4.
Condition on discharge measured by physical signs: Improved, 3; stationary, 1.
Cod-liver oil agreed, 1; disagreed, 3.
Emulsion agreed, 4; disagreed, 0.

6 cases in the 2nd stage—Condition on discharge measured by general symptoms: Improved, 6.
Condition on discharge measured by physical signs: Improved, 6.
Cod-liver oil agreed, 0; disagreed, 6.
Emulsion agreed, 6; disagreed, 0.

6 cases in the 3rd stage—Condition on discharge measured by general symptoms: Improved, 6.
Condition on discharge measured by physical signs: Improved, 5; stationary, 1.
Cod-liver oil agreed, 0; disagreed, 6.
Emulsion agreed, 6; disagreed, 0.

Total results of the 16 cases measured by general symptoms: Improved, 16.
Measured by physical signs: Improved, 14; stationary, 2.
Emulsion agreed, 16; disagreed, 0.
Cod-liver oil agreed, 1; disagreed, 15.

The average quantity of emulsion taken by each patient was about one ounce avoirdupois in one pint of milk each day for eight weeks.

A THIRD REPORT

OF

Cases Treated with Pancreatic Emulsion,

AT THE ROYAL INFIRMARY FOR DISEASES OF THE CHEST, CITY-ROAD.

[From the Lancet, Nov. 11 and 18, 1865.]

In my former reports I have referred to the able assistance I have received, in my experiments on pancreatic juice, from Mr. Heathorn; and I am sorry to have to state that, since my last report of June 10th, Mr. Heathorn has found his other engagements too numerous to allow him to prepare the emulsions, now that the demand has increased through other medical men prescribing them as well as myself.

We have, however, been able to make great improvements in the mode of extracting the active principles of pancreatic juice, and of preparing pancreatic emulsion; and I have recently received the most valuable assistance in this matter from Mr.

Schweitzer, the scientific chemist in the establishment of Messrs. Savory and Moore. In future, therefore, the active principles of pancreatic juice and pancreatic emulsions will be prepared by Messrs. Savory and Moore. I am happy to be able to make this announcement, because it is a guarantee that these delicate remedial agents will be prepared with systematic accuracy, and that every opportunity will be given for their general use by medical men. I have requested Messrs. Savory and Moore to keep the preparations of pancreatic juice in the following forms:—

1. Pancreatic emulsion of solid fat.
2. The active principles of pancreatic juice; to be swallowed soon after a full meal. Each dose to represent a certain emulsifying power.
3. Pancreatic emulsion of cod-liver oil.
4. Pancreatic emulsion of lard oil.

I would suggest to those who have the opportunity of doing so, to try the effects of these preparations in cases of diabetic wasting, and in cases of disease of the pancreas, where the secretion of the organ is arrested or deteriorated; for it is probable that in these cases life may be prolonged by the artificial introduction of pancreatic juice.

My own experience of the use of pancreatic juice

has, at present, been almost confined to the treatment of tuberculosis; and the following is a summary of cases treated with pancreatic emulsions of lard oil and of suet since my last paper:—

Summary of Thirty-eight Cases of Consumption; Twenty-seven treated with Pancreatic Emulsion of Lard Oil, and Eleven with Pancreatic Emulsion of Suet.

FIRST STAGE (ADVANCED).

9 cases in the 1st stage treated with *lard-oil emulsion*—Condition on discharge measured by general symptoms: Improved, 8; worse, 1.

Condition on discharge measured by physical signs: Improved, 5; stationary, 3; worse, 1.

Cod-liver oil agreed, 4; disagreed, 5.

Emulsion agreed, 9; disagreed, 0.

SECOND STAGE.

9 cases treated with *lard-oil emulsion*—Condition on discharge measured by general symptoms: Improved, 8; worse, 1.

Condition on discharge measured by physical signs: Improved, 5; stationary, 2; worse, 2.

5 cases treated with *suet emulsion*—Condition on discharge measured by general symptoms: Improved, 3; stationary, 1; worse, 1.

Condition on discharge measured by physical signs: Improved, 2; stationary, 3.

Cod-liver oil agreed, 5; disagreed, 9.

Emulsion agreed (lard-oil, 8, suet, 5), 13; disagreed (lard-oil), 1.

THIRD STAGE.

9 cases treated with *lard-oil emulsion*—Condition on discharge measured by general symptoms: Improved, 7; worse, 2.

Condition on discharge measured by physical signs: Improved, 3; stationary, 5; worse, 1.

6 cases treated with *suet emulsion*—Condition on discharge measured by general symptoms: Improved, 5; worse, 1.

Condition on discharge measured by physical signs: Improved, 3; stationary, 2; worse, 1.

Cod-liver oil agreed, 5; disagreed, 10.

Emulsion agreed (lard-oil 8, suet 6), 14; disagreed (lard-oil), 1.

Total Results of the Twenty-seven Cases treated with Lard-oil Emulsion —

Measured by general symptoms: Improved, 23; worse, 4.

Measured by physical signs: Improved, 13; stationary, 10; worse, 4.

Total Results of the Eleven Cases treated with Suet Emulsion :—

Measured by general symptoms: Improved, 8; stationary, 1; worse, 2.

Measured by physical signs: Improved, 5; stationary, 5; worse, 1.

Of the total number of 38 cases—

9 were in the first stage (advanced).

14 „ second stage.

15 „ third stage.

Cod-liver oil agreed, 14; disagreed, 24.

Emulsion agreed, 36; disagreed, 2.

The effects of the emulsions on the digestive functions may be partly learned from the following short extracts from some of the cases :—

Case 55.—When he began emulsion, appetite was very bad, and it was difficult to take the milk with emulsion; but appetite improved as he went on, and he took emulsion twenty weeks with great advantage, buying it for himself after his discharge, because he missed it when it was discontinued.

Case 56.—After taking oil for a long while and losing flesh and strength, he gained 8 lb. in seven weeks under emulsion, and got quite strong.

Case 57.—Had taken oil off and on for twelve

months, but steadily lost flesh and strength. Of late oil has disagreed, and motions are pale. Emulsion agrees; appetite improves, and she gains flesh and strength rapidly; motions no longer pale.

Case 59.—Advanced third stage. Had taken cod-liver oil for three months, but it disagreed latterly, and she had steadily lost ground in all respects. Emulsion agrees well, and after a fortnight she is surprised at her improvement. At the fourth week she writes: "I gain strength daily. Appetite much improved. I have not such a dislike for fat, and I now can take cod-liver oil as well as emulsion."

Case 64.—Oil makes him sick. Emulsion keeps down when nothing else will: appetite improves, and he gains flesh, and says he is much stouter and stronger.

Case 62.—Has been ill seven years; is now in the third stage. Has taken cod-liver oil, and he says it agrees and does more good than anything else; but still he gets worse and worse, losing flesh and strength fast. He likes emulsion, and at eighth week he does not lose flesh, and says he feels firmer; appetite better. At twelfth week he still gains ground. Ordered to take cod oil as well as emulsion.

Case 70.—Cod-liver oil agreed last winter, but now won't keep down. Emulsion agrees well.

Case 71.—Has been taking cod-liver oil freely, but it does no good; loses flesh, appetite, and strength. Emulsion agrees well. At sixth week has gained flesh and strength; appetite good.

Case 72.—Neither cod-liver oil, suet and milk, milk, nor eggs will keep down. Emulsion agrees, keeps down, creates appetite for meat; can take milk with the emulsion.

Case 75.—Oil agreed until lately; now it causes sickness and destroys appetite. Emulsion agrees; she likes it; appetite improves; feels less sinking. At sixth week she feels herself quite well.

Case 76.—Oil has been taken five weeks with no good effect, and now it won't keep down. Emulsion agrees; he says he "feels support from it;" appetite improves, and he gains flesh and good looks.

Case 79.—Oil won't keep down. Emulsion agrees, and makes her "feel stronger inside."

Case 81.—Oil keeps down, but passes off by the bowels. Emulsion agrees, and does not pass off by the bowels.

Case 85.—Oil comes up as fast as it goes down. Emulsion agrees well. At sixth week discharged much improved.

Case 86.—Third stage. Oil won't keep down,

though tried many times; it produces violent sickness. Suet and milk without emulsion kept down, but caused "dreadful feeling of sickness." Emulsion of suet in milk keeps down, and produces no feeling of sickness; appetite improves. Discharged at twelfth week very much improved in all respects. After four weeks comes back to beg for more emulsion, saying she can't live without it. A fortnight after this, says emulsion agrees, "keeps down the food and keeps off sickness."

Case 92.—Physical signs of consumption not very marked, but general condition very bad; great prostration and sweating; very troublesome cough. Has had much tonic treatment and cod-liver oil, but has not found anything do good. Emulsion agrees. After fourth week is remarkably improved; says he feels a great vacancy when he does not take the emulsion.

Dr. Moxon, of Kirton-in-Lindsey, writes me as follows of the good effects of the emulsion: "I have tried the pancreatic emulsion in the case of a young man affected with phthisis in quite an early stage. There is tubercle deposited in the apex of one lung. He had lost all appetite, complained of pain in the side, and tightness of the chest. He had only slight cough, great lassitude, and had lost weight con-

siderably. He had been under treatment by Dr. —— without any relief; tonics had not the slightest effect upon him. On seeing your report in *The Lancet*, I determined to try the 'emulsion,' and gave up all other treatment, except the occasional use of a stimulant liniment. He has taken four pounds of the emulsion. He had no difficulty in taking it; but, on the contrary, found it palatable. His appetite has improved, so that he takes food with a good relish. Has gained a little in weight. Does not feel any lassitude, except at times: has lost the sense of constriction in the chest, and is altogether much better, It is now six weeks since he has given it up.... I am very pleased with its effects....I shall certainly use it again when I have a case which requires it."

Dr. Robt. Growse, of Brentwood, writes: "I have employed the pancreatic emulsion simply for the purpose of increasing flesh. It is much more palatable than the cod-liver oil, is well borne by the stomach, and will, I think, materially aid me in the object I have in view."

Dr. J. W. Phillips, of Cowbridge, Glamorganshire, writes that he gave the emulsion to "a young lady, aged eighteen, in the third stage of phthisis. She had failed taking cod-liver oil for some time. After taking the emulsion she recovered considerably, was

able to resume out-door exercise and cod-liver oil. She took in all three pounds."

A remarkable case of recovery has just come to my notice, in which the emulsion appears to have played an important part.

A gentleman consulted me in March last (1865), of whom I made the following note :—Aged thirty; losing flesh fast; getting very weak; sweats much; appetite moderate; cannot touch fat. Cough troublesome all day, and also in the night; now and then the fits end in vomiting; much expectoration, occasionally tinged with blood. Has had a cough for three winters, with bronchitis and frequent catarrh; but has every appearance, now, of rapid phthisis. Chest very thin; supra and infra-clavicular spaces hollow. Left upper lobe dull in front; breathing harsh; expiration long and harsh; no moist sounds. Right upper lobe dull behind; breathing very harsh, especially expiration; no moist sounds; other parts normal. He had been long under treatment for his cough and weakness without benefit, and was fast getting worse. I ordered the pancreatic emulsion of suet, and some other treatment; but at the end of a week he had not improved, and found some difficulty in taking the emulsion. His friends came to me about him in great anxiety, as he seemed to be rapidly sinking.

I advised his removal to the south coast, and that if possible, he should go on with the emulsion. With some difficulty, in consequence of his weakness, this advice was followed. At the end of a week he wrote me that he was now able to take the emulsion once a day, but that he felt getting worse instead of better; and his friends called and reported that they had seen him, and thought he could not possibly recover. I advised that he should put himself under a doctor in the place where he was, who could communicate with me. From this time I heard no more of him; and about a month afterwards filled up my note of his case thus: "From the last reports I fear that he sank rapidly and died." To my great surprise, however, he called upon me last week (Oct. 10th), looking the picture of health, so stout and strong that I did not at first recognise him. He told me that when my last message reached him he was just beginning to mend, and, therefore, did not send for the local doctor; that he had been wandering about the south coast from place to place, and had taken the emulsion once or twice a day regularly for twenty-eight weeks, with no other medical treatment. On examining his chest, I found the physical signs unchanged since my first note; the dulness and harsh

breathing remained, but no moist sounds and no cavities. His own idea was that his recovery was mainly due to the emulsion, because he had found that he missed it so much when he had once or twice been prevented from taking it by the delay of his parcel from London.

July 25th, 1865.—I was consulted about a little girl aged thirteen, chiefly for an opinion as to whether anything more could be done to prolong life. She had been under treatment for several years for scrofulous disease of the right knee, and during that time had taken much cod-liver oil and tonics, from which her general health had been often benefited; the knee had got worse and worse, and all hope of saving the limb had been abandoned for about twelve months by several leading surgeons, and amputation decided on. But the child's health had so completely broken down, and consumption so unmistakably set in, that, after some fruitless attempts to recover her strength, the idea of operating had also been abandoned. When I saw her, she was very thin, careworn and haggard, with hectic flush, loss of appetite, severe night-sweats, constant cough, and considerable expectoration; the upper part of the right lung partially dull, breathing harsh; upper half of the left lung dull, copious

crepitation and bronchophony back and front; the pulse feeble and rapid. I found that she had been steadily taking a tablespoonful of cod-liver oil twice a day for two months, but losing flesh and strength all the time. As a last resource, I ordered the oil to be taken once instead of twice, and the pancreatic emulsion of suet once.

Oct. 17th, 1865.—I was again requested to see the child for the purpose of saying whether she might now have the operation performed, as her parents thought her so much improved in health. I learned that the emulsion and oil had both agreed, and had been taken regularly since my last visit; she was rather tired of the oil, but not of the emulsion. She had gained much flesh and lost the haggard expression. Nightsweats had ceased, and the cough was so nearly well that she did not even require a lozenge. No expectoration; appetite fair. The dulness and bronchophony remained, but no moist sounds could be heard in either lung.*

The length to which this report has already extended prevents me from relating other interesting cases which have occurred in private practice, and

* This little patient improved so much under the continued use of the emulsion, that on Feb. 24th, 1866, she was able to undergo the operation of amputation of the leg above the knee, and she has since made an excellent recovery.

from giving some information which I have obtained regarding the subsequent course of hospital cases mentioned in my former reports.

I am anxious, however, not to close this paper without suggesting a rational explanation of the frequent failure of our attempts to keep up that favourable change which so often occurs in a case of consumption when cod-liver oil is first administered. We all know how constantly it happens that a consumptive patient makes remarkable progress for a certain time while taking cod-liver oil, if it is well digested—a progress which might well lead us to hope that it would end in a cure; and we all know equally well how constantly this progress stops at a certain point, beyond which the recovery does not seem able to advance, and from which it too often happens that, sooner or later, a gradual descent takes place.

The suggestion I wish to make is, that, assuming a defect to exist in the natural power of digesting and assimilating fats, it would be irrational to expect anything else to happen than that which we witness.

According to the careful estimate of Dr. Lyon Playfair,* the quantity of fat required by an adult in twenty-four hours, to keep up healthy nutrition,

* "The Food of Man in Relation to his Useful Work." 1865.

is from 1 oz. to 2·5 oz.; and according to the estimates made from very numerous and carefully-selected data by Mr. Farrants and myself,* the quantity is from 2 oz. to 3·5 oz. We may fairly assume, then, that not less than two ounces of fat per day, on an average, is required to keep up healthy nutrition in an adult. We have next to bear in mind, that before a case of consumption ordinarily attracts attention, and begins to be treated as such, many pounds weight, principally consisting of fat, have been gradually removed from the body. In this condition—1, a deficiency of fat throughout the organism; 2, a loss of the power to assimilate ordinary fats; 3, a constant demand for two ounces per day to maintain healthy nutrition,—we administer cod-liver oil, in the belief that this form of fat will assimilate when other forms will not. Supposing that it agrees, and that some or all of it is utilized, a rapid improvement takes place in the patient, from the supply of some of that for want of which life was steadily fading—very much as a cut flower that has drooped for want of its supply of sap, rallies and recovers freshness for a time when put into water. But there are very few persons who can take more than from half an ounce to one ounce of

* "A Manual of Diet and Regimen." 1864.

oil per day—few who can even take this steadily from week to week without intermissions. But supposing an ounce or an ounce and a half per day to be taken regularly, how is this to supply, not only the two ounces per day required for healthy nutrition, but all the extra ounces of arrears that were lost before the treatment was begun? But assuming the possibility of two ounces per day of oil for nutrition, and another two ounces for arrears, being taken and utilized, even then the *whole thing may be unstable and may break down*, from the fact that we are supplying oil and *not solid fat*—a body rich in olein and poor in stearin and margarin, in the place of bodies rich in stearin and margarin and poor in olein, such as the fats taken in normal food.

The practical conclusion from these considerations appears to be, that if we are to give a fair chance of recovery to a patient deprived of the natural powers of digesting and assimilating fats, we must, by one means or another, secure that two ounces of fat of average solidity are utilized every day for the purposes of nutrition, and an additional ounce or two to make up for arrears.

To obtain this end four principal means are now at our command:—

1st. The administration of as much oil and fat,

either as medicine or food, as the digestive and assimilative powers are still competent to utilize.

2nd. The introduction of fat and oil into the system by rubbing them into the skin of the body and limbs.

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Condition of patients on admission :—

	Cases.
First stage advanced	20
Second stage	34
Third stage	33
Total	87

Condition on discharge measured by general symptoms: Improved, 71; stationary, 3; worse, 10; not noted, 3.

Measured by physical signs: Improved, 45; stationary, 27; worse, 10; not noted, 5.

	Cases.
Emulsion agreed..	82
Cod-liver oil agreed	28
Emulsion disagreed	5
Cod-liver oil disagreed	50
Cod-liver oil not tried	9

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29. First stage of acute bronchitis.
30. First stage of acute pleurisy.
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